```
=> d his

     (FILE 'HOME' ENTERED AT 14:29:07 ON 29 MAR 2001)

     FILE 'CAPLUS, EUROPATFULL, PCTFULL, USPATFULL, MEDLINE, BIOSIS, EMBASE'
     ENTERED AT 14:30:12 ON 29 MAR 2001
L1          7069 S AMLODIPINE OR AMLODIPINE(W)BESYLATE
L2          2370 S ATORVASTATIN OR ATORVASTATIN(3A)HEMICALCIUM
L3          5324 S HYPERTENS?(L)HYPERLIPIDEMI?
L4            37 S L1(L)L3
L5            97 S L2(L)L3
L6            24 S L4(L)L5
```

① indicate which claims will be examined

② modified restriction

1-87 product clai

method clai

32

33

89

90

102(89)

93

78

92

91?

=> d l5 89-93 ibib kwic

L5 ANSWER 89 OF 97 USPATFULL
ACCESSION NUMBER: 2000:128341 USPATFULL
TITLE: Method and pharmaceutical composition for regulating lipid concentration
INVENTOR(S): Bocan, Thomas M. A., Ann Arbor, MI, United States
PATENT ASSIGNEE(S): Warner-Lambert Company, Morris Plains, NJ, United States (U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 6124309	20000926	
	WO 9716184	19970509	
APPLICATION INFO.:	US 1998-51368	19980407	(9)
	WO 1996-US15854	19961002	
		19980407	PCT 371 date
		19980407	PCT 102(e) date

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1995-6155	19951102	(60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Jordan, Kimberly
LEGAL REPRESENTATIVE: Anderson, Elizabeth M.
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 432
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . and those who are at risk of developing various acute ischemic syndromes including individuals with high blood pressure, diabetes, or **hyperlipidemia**, and individuals who smoke.

DETD . . . ischemic syndromes that may be treated by the method of the present invention include: angina pectoris, coronary artery disease (CAD), **hypertension**, cerebrovascular accidents, transient ischemic attacks, chronic obstructive pulmonary disease, chronic hypoxic lung disease, pulmonary **hypertension**, renal **hypertension**, chronic renal disease, microvascular complications of diabetes, and vaso-occlusive complications of sickle cell anemia.

DETD An HMG-COA reductase inhibitor for use in the novel method may be selected from **atorvastatin**, lovastatin, simvastatin, pravastatin, fluvastatin, and rivastatin; preferably **atorvastatin**, lovastatin, or simvastatin; most preferably **atorvastatin**. HMG-COA reductase inhibitors are known to function as antihypercholesterolemic agents. They reduce hepatic cholesterol biosynthesis by inhibiting the enzyme HMG-COA. . . . the early, rate-limiting step in the biosynthesis of cholesterol, the conversion of hydroxymethylglutarate to mevalonate. Known HMG-COA reductase inhibitors include **atorvastatin** MEVACOR.RTM. (lovastatin), ZOCOR.RTM. (simvastatin), PRAVACHOL.RTM. (pravastatin), LESCOL.RTM. (fluvastatin), and rivastatin. ##STR1##

DETD **Atorvastatin** is disclosed in U.S. Pat. No. 5,273,995. Related compounds are disclosed in U.S. Pat. No. 4,681,893.

DETD The lipid modifying and antiatherosclerotic action of

2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sul

famate, **atorvastatin**, and the combination of both compounds was assessed in a rabbit model of atherosclerosis in which the combination of hypercholesterolemia. . .

DETD . . . cholesterol levels and administered the 0% C, 3% PNO, 3% CNO diet either alone or containing N-(2,6-diisopropyl-phenyl)-2-(2-dodecyl-2H-tetrazol-5-yl)-2-phenyl-acetamide at 10 mg/kg, **atorvastatin** at 5 mg/kg, or N-(2,6-diisopropyl-phenyl)-2-(2-dodecyl-2H-tetrazol-5-yl)-2-phenylacetamide +**atorvastatin** at 10+5 mg/kg for the next 8 weeks.

DETD Relative to the untreated, cholesterol-fed control, plasma total cholesterol levels were unchanged by 2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate but reduced 43% and 67% with **atorvastatin** and 2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]-sulfamate+**atorvastatin**, respectively. Associated with the changes in plasma total cholesterol were marked alterations in the plasma lipoprotein distribution.

2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate reduced % VLDL-cholesterol (VLDL-C) and increased % LDL-cholesterol (LDL-C); **atorvastatin** had limited effect; and upon combination treatment % VLDL-C and % LDL-C were reduced, and % HDL-cholesterol was increased.

DETD TABLE I

Lipoprotein Distribution Expressed as Percent of Total Plasma Cholesterol	VLDL	LDL	HDL
Progression Control	16	60	24
2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]-acetyl]sulfamate (10 mg/kg)	5	73	22
Atorvastatin (5 mg/kg)	14	48	38
2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]-acetyl]sulfamate + **Atorvastatin** (10 + 5 mg/kg)	4	35	60

DETD . . . the thoracic aorta; however, the incidence of complex fibrous plaques within the iliac-femoral artery was reduced from 50% to 14%. **Atorvastatin** reduced the CE enrichment of both vascular regions by 27% to 41% without changing the gross extent of thoracic lesions and incidence of fibrous plaques.

2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate+**atorvastatin** had no effect on the CE enrichment of the thoracic aorta and gross extent of thoracic aortic lesions; however, the. . . plaques was decreased to 17%. Comparison of the data relative to the time zero control, i.e., prior to drug administration, **atorvastatin** alone and in

combination with 2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate significantly reduced the CE enrichment of the iliac-femoral artery. Morphometric analysis of the iliac-femoral artery revealed that **atorvastatin** reduced the lesion size, while the combination of **atorvastatin** and 2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate significantly decreased the monocyte-macrophage content of the lesion without changing lesion size. 2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate alone had no effect on. . .

DETD Therefore, it is clear that a combination of N-(2,6-diisopropyl-phenyl)-2-(2-dodecyl-2H-tetrazol-5-yl)-2-phenyl-acetamide and **atorvastatin** administered in a chow/fat diet results in a greater reduction in plasma apo B-containing lipoprotein than either alone and that a normalization of the plasma lipoprotein distribution is achieved. **Atorvastatin** not only blunts the cholesteryl ester enrichment of the vasculature but also decrease the lipid enrichment of a pre-existing atherosclerotic lesion. 2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate+ **atorvastatin** reduces the CE enrichment of pre-existing atherosclerotic lesions to the same extent as **atorvastatin** alone, but the atherosclerotic lesions are less complicated with respect to their histologic character.

L5 ANSWER 90 OF 97 USPATFULL

ACCESSION NUMBER:	2000:95018 USPATFULL	
TITLE:	Method and pharmaceutical composition for regulating lipid concentration	
INVENTOR(S):	Bocan, Thomas M. A., Ann Arbor, MI, United States	
PATENT ASSIGNEE(S):	Warner-Lambert Company, Morris Plains, NJ, United States (U.S. corporation)	

	NUMBER	DATE	
PATENT INFORMATION:	US 6093719	20000725	
APPLICATION INFO.:	US 1999-345944	19990701	(9)
RELATED APPLN. INFO.:	Division of Ser. No. US 51368		

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1995-6155	19951102	(60)

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Jordan, Kimberly
LEGAL REPRESENTATIVE:	Anderson, Elizabeth M.
NUMBER OF CLAIMS:	3
EXEMPLARY CLAIM:	1
LINE COUNT:	409

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . and those who are at risk of developing various acute ischemic syndromes including individuals with high blood pressure, diabetes, or **hyperlipidemia**, and individuals who smoke.

DETD . . . ischemic syndromes that may be treated by the method of the present invention include: angina pectoris, coronary artery disease (CAD), **hypertension**, cerebrovascular accidents, transient ischemic attacks, chronic obstructive pulmonary disease, chronic hypoxic

lung disease, pulmonary **hypertension**, renal **hypertension**, chronic renal disease, microvascular complications of diabetes, and vaso-occlusive complications of sickle cell anemia.

DETD An HMG-CoA reductase inhibitor for use in the novel method may be selected from **atorvastatin**, lovastatin, simvastatin, pravastatin, fluvastatin, and rivastatin; preferably **atorvastatin**, lovastatin, or simvastatin; most preferably **atorvastatin**.

DETD . . . the early, rate-limiting step in the biosynthesis of cholesterol, the conversion of hydroxymethylglutarate to mevalonate. Known HMG-CoA reductase inhibitors include **atorvastatin** MEVACOR.RTM. (lovastatin), ZOCOR.RTM. (simvastatin), PRAVACHOL.RTM. (pravastatin), LESCOL.RTM. (fluvastatin), and rivastatin. ##STR1##

DETD **Atorvastatin** is disclosed in U.S. Pat. No. 5,273,995. Related compounds are disclosed in U.S. Pat. No. 4,681,893.

DETD The lipid modifying and antiatherosclerotic action of

2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate, **atorvastatin**, and the combination of both compounds was assessed in a rabbit model of atherosclerosis in which the combination of hypercholesterolemia. . .

DETD . . . cholesterol levels and administered the 0% C, 3% PNO, 3% CNO diet either alone or containing

N-(2,6-diisopropyl-phenyl)-2-(2-dodecyl-2H-tetrazol-5-yl)-2-phenyl-acetamide at 10 mg/kg, **atorvastatin** at 5 mg/kg, or N-(2,6-diiso-propyl-phenyl)-2-(2-dodecyl-2H-tetrazol-5-yl)-2-phenyl-acetamide+**atorvastatin** at 10+5 mg/kg for the next 8 weeks.

DETD Relative to the untreated, cholesterol-fed control, plasma total cholesterol levels were unchanged by

2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate but reduced 43% and 67% with **atorvastatin** and 2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]-sulfamate+**atorvastatin**, respectively. Associated with the changes in plasma total cholesterol were marked alterations in the plasma lipoprotein distribution.

2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate reduced % VLDL-cholesterol (VLDL-C) and increased % LDL-cholesterol (LDL-C); **atorvastatin** had limited effect; and upon combination treatment % VLDL-C and % LDL-C were reduced, and % HDL-cholesterol was increased.

DETD . . . of Total Plasma Cholesterol

	VLDL	LDL	HDL
Progression Control	16	60	24
2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]-acetyl]sulfamate (10 mg/kg)	5	73	22
Atorvastatin (5 mg/kg)	14	48	38
2,6-bis(1-methylethyl)-phenyl[[2,4,6-tris(1-methylethyl)phenyl]-acetyl]sulfamate + **Atorvastatin** (10 + 5 mg/kg)	4	35	60

DETD . . . the thoracic aorta; however, the incidence of complex fibrous plaques within the iliac-femoral artery was reduced from 50% to 14%. **Atorvastatin** reduced the CE enrichment of both vascular regions by 27% to 41% without changing the gross extent of thoracic lesions and incidence of fibrous plaques.

2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate+**atorvastatin** had no effect on the CE enrichment of the thoracic aorta and gross extent of thoracic aortic lesions; however, the. . . plaques was decreased to 17%. Comparison of the data relative to the time zero control, i.e., prior to drug administration, **atorvastatin** alone and in combination with 2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)phenyl]acetyl]sulfamate significantly reduced the CE enrichment of the iliac-femoral artery Morphometric analysis of the iliac-femoral artery revealed that **atorvastatin** reduced the lesion size, while the combination of **atorvastatin** and 2,6-bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate significantly decreased the monocyte-macrophage content of the lesion without changing lesion size. 2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate alone had no effect on. . .

DETD Therefore, it is clear that a combination of N-(2,6-diisopropyl-phenyl)-2-(2-dodecyl-2H-tetrazol -5-yl)-2-phenyl-acetamide and **atorvastatin** administered in a chow/fat diet results in a greater reduction in plasma apo B-containing lipoprotein than either alone and that a normalization of the plasma lipoprotein distribution is achieved **Atorvastatin** not only blunts the cholesteryl ester enrichment of the vasculature but also decrease the lipid enrichment of a pre-existing atherosclerotic lesion. 2,6-Bis(1-methylethyl)phenyl[[2,4,6-tris(1-methylethyl)-phenyl]acetyl]sulfamate+ **atorvastatin** reduces the CE enrichment of pre-existing atherosclerotic lesions to the same extent as **atorvastatin** alone, but the atherosclerotic lesions are less complicated with respect to their histologic character.

L5 ANSWER 91 OF 97 USPATFULL

ACCESSION NUMBER:	2000:34393 USPATFULL
TITLE:	Systemic inflammatory markers as diagnostic tools in the prevention of atherosclerotic diseases and as tools to aid in the selection of agents to be used for the prevention and treatment of atherosclerotic disease
INVENTOR(S):	Ridker, Paul, Chestnut Hill, MA, United States Hennekens, Charles H., South Natick, MA, United States
PATENT ASSIGNEE(S):	The Brigham and Women's Hospital, Inc., Boston, MA, United States (U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 6040147	20000321	
APPLICATION INFO.:	US 1998-54212	19980402	(9)
DOCUMENT TYPE:	Utility		
PRIMARY EXAMINER:	Saunders, David		
LEGAL REPRESENTATIVE:	Wolf, Greenfield & Sacks, PC		
NUMBER OF CLAIMS:	47		

102(e)

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1501
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . populations are casual or due to short-term inflammatory changes, or to interrelations with other risk factors, in particular smoking and **hyperlipidemia**.

DETD Lipid reducing agents include gemfibrozil, cholystyramine, colestipol, nicotinic acid, probucol lovastatin, fluvastatin, simvastatin, **atorvastatin**, pravastatin, cirivastatin.

DETD . . . logistic regression models accounting for the matching variables and controlling for randomized treatment assignment, body mass index, diabetes, history of **hypertension**, and a parental history of coronary artery disease. Similar models were employed to adjust for measured baseline levels of total. . .

DETD . . . subsequently developed myocardial infarction were more likely than those who remained free of vascular disease to have a history of **hypertension**, **hyperlipidemia**, or a parental history of coronary artery disease. Similarly, those who subsequently developed stroke were more likely to be **hypertensive**. Due to the matching, age and smoking were similar in cases and controls.

DETD . . . 3.3 25 +/- 3.2 26 +/- 2.9 (kg/m2*)

History of high 9 13 17 10 7 cholesterol (%)
History of **Hypertension** 16 29 27 35 20 (%)
Parental history of 10 13 17 11 8 coronary artery disease (%)

---

*values represent. . .

DETD . . . relationship between C-reactive protein and myocardial infarction was not significantly altered in analyses which adjusted for body mass index, diabetes, **hypertension**, a family history of premature coronary artery disease, total cholesterol, HDL cholesterol, triglycerides, lipoprotein(a), tPA antigen, D-dimer, fibrinogen, or homocysteine. . .

DETD . . . 2.9 0.01
95% CI -- 1.1-4.7 1.0-4.4 1.4-5.9
p -- 0.04 0.04 0.005
Body mass index (kg/m.sup.2), diabetes, history of **hypertension**, and family history of premature CAD
Adjusted RR 1.0 1.5 2.4 2.6 <0.001
95% CI -- 0.9-2.5 1.5-4.0 1.6-4.4
p. . .

DETD . . . not significantly altered in analysis which adjusted for body mass index, diabetes, a family history of premature coronary artery disease, **hyperlipidemia**, and a history of **hypertension**
.

DETD . . . *Matched for smoking and age, controlled for total and HDL cholesterol
Matched for smoking and age, controlled for history of **hypertension, **hyperlipidemia**, body mass index, diabetes, and a family history of premature CAD
95% CI = 95 percent confidence interval

L5 ANSWER 92 OF 97 USPATFULL
ACCESSION NUMBER: 1999:170629 USPATFULL
TITLE: Arylthiazolidinedione derivatives
INVENTOR(S): Sahoo, Soumya P., Old Bridge, NJ, United States
Tolman, Richard L., Los Altos, CA, United States
Han, Wei, West Chester, PA, United States
Bergman, Jeffrey, Tenafly, NJ, United States
Santini, Conrad, Warren, NJ, United States
Lombardo, Victoria K., Belle Mead, NJ, United States
Desai, Ranjit, Franklin Park, NJ, United States
Boueres, Julia K., Franklin Park, NJ, United States
Gratale, Dominick F., Edison, NJ, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6008237	19991228
APPLICATION INFO.:	US 1998-213542	19981217 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-68271	19971219 (60)
	US 1998-105238	19981022 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Gerstl, Robert
LEGAL REPRESENTATIVE: McGinnis, James L.; Rose, David L.; Yang, Mollie M.
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 3470
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . associated with increased and premature mortality due to an increased risk for microvascular and macrovascular diseases, including nephropathy, neuropathy, retinopathy, **hypertension**, stroke, and heart disease. Therefore, control of glucose homeostasis is a critically important approach for the treatment of diabetes.
SUMM **Hyperlipidemia** is a condition which is characterized by an abnormal increase in serum lipids, such as cholesterol, triglycerides and phospholipids. These. . .
SUMM One form of **hyperlipidemia** is hypercholesterolemia, characterized by the existence of elevated LDL cholesterol levels. The initial treatment for hypercholesterolemia is often to modify. . .
SUMM . . . the enzymes of the beta-oxidation cycle. Compounds of this group include but are not limited to the fibrate class of **hyperlipidemic** drugs, herbicides and phthalate plasticizers. Peroxisome proliferation is also triggered by dietary or physiological factors such as a high-fat diet. . .
SUMM . . . dual agonists of the .alpha./.gamma. subtypes. These compounds are therefore useful in the treatment, control or prevention of diabetes, hyperglycemia, **hyperlipidemia** (including

hypercholesterolemia and hypertriglyceridemia), atherosclerosis, obesity, vascular restenosis, and other PPAR .alpha., .delta. and/or .gamma. mediated diseases, disorders and conditions.

SUMM . . . in treating, controlling or preventing include, but are not limited to, (1) A diabetes mellitus, (2) hyperglycemia, (3) obesity, (4) **hyperlipidemia**, (5) hypertriglyceridemia, (6) hypercholesterolemia (including raising HDL levels), (7) atherosclerosis, (8) vascular restenosis, (9) irritable bowel syndrome, (10) pancreatitis, (11). . .

SUMM . . . dual agonist is administered with a cholesterol biosynthesis inhibitor, particularly an HMG-CoA reductase inhibitor such as lovastatin, simvastatin, pravastatin, fluvastatin, **atorvastatin** and rivastatin.

SUMM (e) cholesterol lowering agents such as (i) HMG-CoA reductase inhibitors (lovastatin, simvastatin and pravastatin, fluvastatin, **atorvastatin**, rivastatin and other statins), (ii) sequestrants (cholestyramine, colestipol and a dialkylaminoalkyl derivatives of a cross-linked dextran), (iii) nicotinyl alcohol, nicotinic. . .

L5 ANSWER 93 OF 97 USPATFULL

ACCESSION NUMBER:	1999:166969 USPATFULL
TITLE:	Method of treating hyperlipidemia
INVENTOR(S):	Cawthorne, Michael Anthony, Horsham, United Kingdom Liu, Yong-Ling, Buckingham, United Kingdom Sennitt, Matthew V., Chipstead, United Kingdom
PATENT ASSIGNEE(S):	Biomeasure, Incorporated, Milford, MA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6004928	19991221
APPLICATION INFO.:	US 1998-78111	19980513 (9)
PRIORITY INFORMATION:	US 1997-46346	19970513 (60)

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Russel, Jeffrey E.
LEGAL REPRESENTATIVE:	Conway, John D.Fish & Richardson
NUMBER OF CLAIMS:	23
EXEMPLARY CLAIM:	1
LINE COUNT:	584

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . role of plasma lipids and lipoproteins in atherogenesis (Adult Treatment Panel II, Circulation 89:1333-1445 (1994); Havel, R. J., Clin. Exp. **Hypertens**. 11:887-900 (1989)). Atherogenesis is the process by which lipids accumulate in the intimal lining of arteries leading to the formation. . . metabolism, coagulation, hyperinsulinism and glycation all seem to contribute significantly to the process (Bierman, E. L., Arterio. Throm. 12:647-656 (1992)). **Hyperlipidemia**'s characteristics of raised plasma concentrations of triglyceride, raised low density lipoprotein (LDL) cholesterol concentrations, and low concentrations of high density. . . M, et al., N. Engl. J. Med. 334:374-381 (1996); and Hamsten, A., et al., N. Engl. J. Med. 313:1557-1563 (1985)). **Hyperlipidemia** in

clinical practice, defined by the upper 10 percent of the distribution of plasma lipid levels in a population, i.e.,. . . cholesterol and triacylglycerides in the plasma have become widespread in clinical practice which permits the identification of patients with asymptomatic **hyperlipidemia**. Guidelines are available for diagnosis and monitoring responses to therapy. See Workshop Treatment of **Hyperlipidemia**, 1996-2 (Lakesmedelsverket, Uppsala, Sweden 1996). Lowering plasma lipid concentrations reduces the amount of atherogenic plaques on the intima of blood. . .

SUMM A number of disorders are associated with **hyperlipidemia**, such as uncontrolled diabetes mellitus (insulin-dependent diabetes mellitus and non-insulin-dependent diabetes mellitus) (Bianchi, R., et al., Diab.

Nutr. Metabl. 7:43-51. . . Principles of Internal Medicine, Ed. Braunwald, E., et al., 11th Edition, McGraw-Hill 1016-1024 (1988)). A number of drugs also produce **hyperlipidemia**, such as oral contraceptives, estrogens, glucocorticoids and antihypertensives. Dietary factors such as increased caloric intake (recent weight gain), consumption of foods high in saturated fats and cholesterol and alcohol intake contribute to the development of **hyperlipidemia**. Aside from these, primary **hyperlipidemia** include a family of genetic disorders associated with family histories of **hyperlipidemia** or xanthomas and pancreatitis.

SUMM The present invention relates to a method of treating **hyperlipidemia** in a patient (e.g., a mammal such as a human). The method includes the step of administering a therapeutically effective. . . e.g., administered intravenously, subcutaneously, or by implantation of a sustained release formulation. In one embodiment, the patient is suffering from **hyperlipidemia** (e.g., abnormally high levels of cholesterol, triacylglycerols, or glycerol) and/or is a diabetic (i.e., type-I or type-II diabetic).

SUMM . . . triglycerides, cholesterol, or glycerol, such as fibrates (e.g., bezafibrate, gemfibrozil, and clofibrate), HMG-COA reductase inhibitors (e.g., pravastatin, simvastatin, and fluorastatin, **Atorvastatin**, and Lovastatin), bile acid binding resins (e.g., cholestyramine and colestipol), nicotinic acid compounds (e.g., nicotinic acid and niceritrol), and fish oils. See Workshop Treatment of

Hyperlipidemia 1996-2 (Lakemedelsverket, Uppsala, Sweden, 1996).